Cognitive decline and neurodegeneration in type 2 diabetes: new pathophysiological mechanism and therapeutic strategies

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Metabolic diseases including obesity and type 2 diabetes are associated with cognitive decline and with increased risk of dementia. Studies in genetic models of type 2 diabetes showed a decrease in synaptic function and cognitive decline, effects prevented by chronic caffeine consumption. Moreover, the carotid bodies (CBs), peripheral chemoreceptors classically defined as O2 sensors, are also involved in energy and glucose homeostasis, being its dysfunction involved in the development of dysmetabolic states. In fact, the abolishment of CBs activity, via the resection or neuromodulation of its sensitive nerve, the carotid sinus nerve (CSN), prevents and reverses dysmetabolic features in prediabetes and T2D animal models. Additionally, it has been shown that caffeine modulated CB function in control and pathological conditions.
In the present talk I will present new data showing that both chronic caffeine intake and abolishment of CB activity restore short-term memory and cognitive performance in dysmetabolic rats and will explore the mechanisms behind these effects.

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